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# THE REACTION OF THE $F^2$ GENERATION OF A CROSS BETWEEN A COMMON AND A DURUM WHEAT TO TWO BIOLOGIC FORMS OF PUCCINIA GRAMINIS

G. F. PUTTICK[1]

For many years efforts have been made to develop by hybridization varieties of wheat resistant to *Puccinia graminis tritici* Erikss. & Henn. It was generally supposed that only one form of the rust commonly attacked wheat. However, Stakman and Piemeisel (9), Melchers and Parker (5, 6), Levine and Stakman (4) and Stakman, Levine and Leach (11) have shown that in reality *P. graminis tritici* consists of several biologic forms. Each biologic form can infect only certain varieties. No known form can attack all varieties of *Triticum sp.* so far tested; and no variety of any species of Triticum is resistant to all biologic forms, except Khapli (C. I. 4103), a variety of *T. dicoccum*.[2]

Obviously, therefore, it is necessary to breed varieties resistant to several or all of the biologic forms which attack wheat, if the work is to be of practical value.

Previous to the discovery of these biologic forms, Biffen (1, 2) and Nilsson-Ehle (7), had shown that resistance and susceptibility to *Puccinia glumarum* E. & H. segregate in the progeny of crosses according to Mendelian laws. More recently, Hayes, Parker and Kurtzweil (3) have shown that resistance and susceptibility to a single biologic form of *P. graminis* segregate differently in different crosses. But no study

[1] Published, with the approval of the Director, as Paper 245 of the Journal Series of the Minnesota Agricultural Experiment Station.

The work was done under the direction of Prof. E. C. Stakman and Prof. H. K. Hayes, to whom thanks are due for helpful suggestions and criticisms.

The crosses were made by Prof. H. K. Hayes and the $F_1$ generation was grown in the plant breeding nursery in 1919. There was a heavy infection of rust in the breeding plots in 1919 and the $F_1$ proved susceptible to the form or forms of rust present.

The biologic forms used were isolated by Stakman and Levine and had been studied for some time.

[2] Unpublished data furnished by Stakman and Levine.

has been made of the inheritance of resistance and susceptibility in the progeny of crosses between parents which react differently towards several biologic forms of rust. The writer therefore has attempted to determine the reaction of the $F_2$ plants of a cross between two varieties of wheat which react reciprocally to two biologic forms of stem rust.

## MATERIALS AND METHODS

Each plant of the $F_2$ generation from crosses between Mindum (Minn. No. 470), a selection from Arnautka (*Triticum durum*), and Marquis, a common spring wheat (*T. vulgare*) was inoculated with two biologic forms of stem rust.

The first rust form used was collected in November, 1918, at Pocatello, Idaho. Since then it has been cultured on many wheat varieties. These were used as differential hosts in the investigations of Stakman and Levine on biologic specialization. Approximately 20 generations of urediniospores have been cultured. All trials showed that this rust remains constant in its parasitic capabilities. It attacks Mindum normally, whereas Marquis, the other parent of the cross, is highly resistant. This biologic form will be referred to as Form 19.[3]

The second biologic form was collected at St. Paul, Minn., in June, 1918. It had been cultured through approximately 20 urediniospore generations and there was no indication of any change in its parasitic capabilities. In contrast to Form 19, it attacks Marquis severely, whereas Mindum is highly resistant or immune. It will be referred to as Form 1.

The $F_2$ plants were grown in six inch pots in the greenhouse, 5 plants to each pot. When the plants were about three inches high they were inoculated with Form 19. The methods used were essentially the same as those described by Stakman and Piemeisel (8).

Notes on the character of infection with both forms were taken from 14 to 20 days after the date of inoculation. The amount and rapidity of rust development always varies in response to differences in environmental conditions. However, the character of infection always remains the same, within certain narrow limits, regardless of external environment and the age of the plant.

When notes on the character of infection induced by Form 19 had been recorded, the infected leaves were cut off, and the plants were sprayed with water to remove any spores which may have lodged on

[3] The numbers used to designate the biologic forms are the same as those used by Stakman and Levine.

them. The same plants were then inoculated with Form 1. After the regular period of incubation, they were placed on a greenhouse bench sufficiently far removed from the cultures of Form 19 to prevent contamination. Since all the families were not inoculated at the same time, 10 to 12 check plants of both parents were inoculated at the same time as each lot of $F_2$ plants.

The percentage of infection obtained with Form 19 was considerably higher than that obtained with Form 1. It repeatedly was observed that a film of moisture had not been deposited on the leaves of plants placed in the incubation chambers This difficulty was largely overcome by atomizing the plants with water, three times a day, while they were still in the incubation chambers.

In recording the type and degree of infection, the symbols prepared by Stakman and Levine in their work on biologic specialization were used. (See table 1.)

TABLE 1.

*Explanation of symbols used to indicate types and degrees of infection of wheat varieties by Puccinia graminis*

0. IMMUNE
   No uredinia developed; hypersensitive flecks sometimes present.
1. VERY RESISTANT
   Uredinia minute and isolated; surrounded by sharp, continuous, hypersensitive areas.
2. MODERATELY RESISTANT
   Uredinia isolated and small to medium in size; hypersensitive areas present; pustules often surrounded by green islands.
3. MODERATELY SUSCEPTIBLE
   Uredinia medium in size; coalescence infrequent; development of rust somewhat subnormal; true hypersensitiveness absent; chlorotic areas, however, may be present.
4. VERY SUSCEPTIBLE
   Uredinia large, numerous and confluent; hypersensitiveness entirely absent.

These limits are sufficiently narrow to make it possible always to refer the type of infection to one of the established classes, the fluctuations being indicated by the addition of plus or minus signs to the number representing the class.

## EXPERIMENTAL DATA

Table 2 summarizes the results obtained from inoculations with Form 19. Marquis and Mindum were inoculated as controls. On Marquis the type of infection ranged from 1– to 1+ and on Mindum it varied from 3 to 4–. In the same table the $F_2$ plants are classified according

to types of infection. All types of infection appeared. Some plants were immune, while others were completely susceptible. Between these extremes, the reaction of the plants intergraded almost imperceptibly. The parental types reappeared while some were more resistant than the Marquis parent, and others were more susceptible than the Mindum parent.

TABLE 2.

*Type of infection caused by Form 19 on the Marquis and Mindum controls and the distribution of $F_2$ individuals in the various classes of infection*

| PARENTS OR CROSSED FAMILIES | PERIOD OF RUST DEVELOPMENT (DAYS) | PER CENT INFECTION | CLASSES OF INFECTION | | | | | | | | | | | | | | TOTAL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | 0 | 0; | 1– | 1 | 1+ | 2– | 2 | 2+ | 3– | 3 | 3+ | 4– | 4 | 4+ | |
| Parents | | | | | | | | | | | | | | | | | |
| Marquis | 16 | 84 | 23 | | 8 | 94 | 16 | | | | | | | | | | 141 |
| Mindum | 16 | 88 | 18 | | | | | | | | | 53 | 27 | 41 | | | 139 |
| Crossed | | | | | | | | | | | | | | | | | |
| Family 20 | 19 | 92 | 11 | 10 | 9 | 8 | 1 | 9 | 11 | 11 | 9 | 13 | 1 | 1 | 5 | 7 | 106 |
| " 5 | 17 | 76 | 15 | 1 | 2 | 8 | 2 | 4 | 7 | 3 | 7 | 5 | 1 | | 2 | 5 | 62 |
| " 6 | 19 | 73 | 16 | 2 | 3 | 6 | 3 | 3 | 2 | 4 | 6 | 7 | 1 | | 2 | 5 | 60 |
| " 4 | 17 | 56 | 17 | | 1 | 4 | 1 | 3 | 1 | | 5 | 1 | | | 5 | 1 | 39 |
| " 3 | 17 | 53 | 24 | 2 | | 3 | | 3 | | | 5 | 7 | 2 | | 3 | 2 | 51 |
| " 30 | 16 | 89 | 8 | 3 | 3 | 7 | 2 | 7 | 9 | 8 | 5 | 7 | 2 | | 7 | 4 | 72 |
| " 13 | 17 | 90 | 7 | 8 | 6 | 8 | 1 | 7 | 3 | 6 | 4 | 9 | 1 | | 5 | 7 | 72 |
| " 15 | 14 | 84 | 15 | 2 | 2 | 4 | 1 | 3 | 9 | 15 | 4 | 17 | 8 | | 4 | 9 | 93 |
| " 25 | 18 | 93 | 5 | 3 | 6 | 8 | 1 | 1 | 4 | 4 | 14 | 3 | 6 | 2 | 8 | 8 | 73 |
| " 37 | 14 | 77 | 12 | 5 | | 9 | 2 | 2 | 3 | 3 | 3 | 3 | 3 | | 2 | 4 | 51 |
| " 35 | 16 | 75 | 15 | 2 | 2 | 3 | 1 | | 2 | 6 | 9 | 6 | 3 | 3 | 1 | 8 | 61 |
| " 11 | 16 | 87 | 11 | | 3 | 1 | 6 | 6 | 11 | 9 | 5 | 15 | 2 | 2 | 4 | 4 | 85 |
| " 34 | 16 | 98 | 1 | 5 | 5 | 7 | 4 | 1 | | 2 | 6 | 4 | 3 | 1 | 1 | 2 | 42 |
| " 36 | 15 | 99 | 1 | 6 | 20 | 9 | 2 | 2 | 5 | 6 | 4 | 9 | 3 | | 7 | 7 | 81 |
| " 1 | 15 | 92 | 1 | 1 | 4 | 1 | | | | | 2 | 1 | | | 2 | | 12 |
| 2 | 14 | 69 | 4 | | | 3 | | | 1 | 1 | 1 | 1 | 1 | 1 | | | 13 |
| Total | | | 163 | 50 | 66 | 89 | 27 | 51 | 74 | 78 | 89 | 108 | 37 | 10 | 58 | 73 | 973 |
| Summarized total* | | | | | 232 | | | | | | | 578 | | | | | |
| Ratio | | | | | 1.1±.04 | | | | | | | 2.9±.04 | | | | | |

* Total, showing plants as resistant as Marquis, or more so, and those in the intermediate and susceptible classes.

A total of 973 plants were inoculated and no sign of infection appeared on 163 of these. The remaining 810 plants were distributed somewhat irregularly in the various classes of infection. As forms were

obtained which are more resistant than Marquis and others which are more susceptible than Mindum, as well as all classes of intermediates, it seems evident that the parents differ by more than a single genetic factor. When all of the intermediate and susceptible plants are compared with those as resistant as or more resistant than Marquis, a ratio of $2.9 \pm .04 : 1.1 \pm .04$ results. While this grouping of plants without a breeding test in the $F_3$, is not a very accurate means of determining the factor relations, the possibility of a single main factor which governs susceptibility and resistance in the parents, and modifying factors which lead to various degrees of infection, are suggested by the results.

TABLE 3.

*Type of infection caused by Form 1 on the Marquis and Mindum controls and the distribution in the various classes of infection of $F_2$ plants*

| PARENTS OR CROSSED FAMILIES | PERIOD OF RUST DEVELOPMENT | PERCENTAGE OF PLANTS INFECTED | CLASSES OF INFECTION OF $F_2$ PLANTS. | | | | | | | | | | | | | | TOTAL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | 0 | 0; | 1– | 1 | 1+ | 2– | 2 | 2+ | 3– | 3 | 3+ | 4– | 4 | 4+ | |
| Parents | | | | | | | | | | | | | | | | | |
| Marquis | 17 | 85 | 20 | | | | | | | | | | | | 8 | 104 | 132 |
| Mindum | 17 | 76 | 33 | 53 | 49 | 4 | | | | | | | | | | | 139 |
| Crossed | | | | | | | | | | | | | | | | | |
| Family 37 | 16 | 67 | 13 | 4 | | 11 | 1 | 1 | 2 | 2 | 1 | 6 | 2 | | 8 | 1 | 52 |
| " 13 | 20 | 72 | 20 | 10 | 5 | 8 | 2 | | 5 | 3 | 2 | 5 | 1 | 1 | 7 | 3 | 72 |
| " 30 | 16 | 71 | 21 | 15 | 5 | 8 | 4 | 3 | 5 | 1 | 3 | | 2 | | 2 | 3 | 72 |
| " 11 | 17 | 88 | 10 | 14 | 4 | 1 | 1 | 4 | 12 | 7 | 7 | 8 | 1 | 2 | | 4 | 75 |
| " 35 | 22 | 69 | 19 | 10 | 8 | | 1 | 4 | 3 | 2 | 3 | 2 | 1 | 2 | 2 | 4 | 61 |
| Total | | | 83 | 53 | 22 | 28 | 9 | 12 | 27 | 15 | 16 | 21 | 7 | 5 | 19 | 15 | 332 |
| 15 | 19 | 35 | 62 | 10 | 2 | 7 | 2 | | 3 | | 1 | 2 | 2 | | 1 | 1 | 93 |
| 6 | 20 | 30 | 42 | 7 | 1 | 4 | | 2 | | | | 1 | 2 | | 1 | | 60 |
| 5 | 17 | 33 | 41 | 7 | 1 | 5 | 1 | 2 | | 1 | 1 | 1 | | | 1 | 1 | 62 |
| 20 | 19 | 27 | 68 | 12 | | 12 | | 1 | 3 | 2 | | | | | 4 | 3 | 107 |
| Total | | | 213 | 36 | 4 | 28 | 3 | 5 | 6 | 3 | 2 | 4 | 6 | | 7 | 5 | 322 |
| 4 | 17 | 49 | 20 | 4 | 1 | 3 | | | 2 | | 2 | 1 | | | 5 | 1 | 39 |
| 3 | 17 | 55 | 9 | 1 | 3 | 4 | | 1 | 4 | 1 | | 1 | 2 | 1 | 1 | 9 | 37 |
| Total | | | 29 | 5 | 4 | 7 | | 1 | 6 | 1 | 2 | 2 | 2 | 1 | 6 | 10 | 76 |
| 1 | 14 | 75 | 3 | 5 | 1 | 1 | | 1 | | | | 1 | | | | | 12 |
| 2 | 14 | 31 | 9 | 2 | | | | | 2 | | | | | | | | 13 |
| Total | | | 12 | 7 | 1 | 1 | | 1 | 2 | | | 1 | | | | | 25 |
| Total | | | 337 | 101 | 31 | 64 | 12 | 19 | 41 | 19 | 20 | 28 | 15 | 6 | 32 | 30 | 755 |

The appearance of sterility in the $F_1$ and the failure of some seeds to germinate eliminated certain plants. Further, some natural crossing in the field probably took place. The extent to which these three factors

have affected the distribution of $F_2$ plants in the various classes of infection is not known.

The results of the inoculations with Form 1 are given in table 3. The severity of infection on most of the Marquis controls was 4+. The Mindum controls were either decidedly resistant ( 1 to 1− ) or immune (0). Those $F_2$ families with the highest percentage of infection have been grouped in table 3. The type of infection on the parents varied within very narrow limits. On the $F_2$ progeny, on the other hand, the type of infection varied between the extreme limits of infection on the parents, in a closely intergrading series. There is evidence, therefore, that several factors are concerned in susceptibility and resistance to Form 1. The small number of individuals infected no doubt has had considerable effect upon the results. Further, some difficulty was experienced in classifying older plants. Uninfected plants often developed hypersensitive flecks, similar to those on immune plants. These may have been placed in the immune class. This might explain the preponderance of plants in the immune class (0).

Although usually only one type of infection occurred on a single plant, more than one appeared on nine individuals. This might possibly have been due to accidental infection with another rust form but it is improbable, because, when the parents were inoculated with spores from each type of uredinium, the resulting infection was uniform. This variation in the type of infection could be due either to environment or to a difference in the parasitic capabilities of urediniospores belonging to a single biologic form.

### COMBINATIONS OF SUSCEPTIBILITY AND RESISTANCE OF $F_2$ PLANTS TO FORM 19 AND FORM 1.

Since data were obtained on the reaction of each of the $F_2$ plants to two biologic forms, it has been possible to study the various combinations of susceptibility and resistance and the numerical proportions in which they occurred. For each family a table was prepared showing the number of plants resistant to both forms, those susceptible to both forms, and those susceptible to one form but resistant to the other. These data have been summarized in table 4. All combinations of susceptibility and resistance to the two biologic forms appeared. Some plants were resistant to both forms, some were susceptible to both, others were resistant to one form and susceptible or intermediate to the other, and still others were intermediate to both forms. Out of a total of 388 plants, 35 were highly resistant to both forms of rust. A ratio of 14.6:1.4 is obtained by combining plants which were as resistant as Marquis to Form 19 and those as resistant as Mindum to Form 1. The ratio might be considered evidence for the belief that resistance to

the two forms of rust depends upon the recombination of two independently inherited factors. If this assumption were correct one of these factors would condition the reaction of the plants to Form 1, while the other would condition their reaction to Form 19. On the other hand, the segregation for either form, when considered independently, shows that several factors are involved.

TABLE 4.

*The reaction of the $F_2$ plants to both biologic forms used*

| *Types of infection obtained with Form 19** | *Types of infection obtained with Form 1** | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0§ | 1– | 1 | 1+ | 2– | 2 | 2+ | 3– | 3 | 3+ | 4– | 4 | 4+ | Total |
| 0 | 13 | 3 | 3 | 1 | | | | 1 | 1 | 1 | | 1 | | 24 |
| 1– | 8† | 2 | | 1 | | 3 | | | 1 | 1 | | | | 16 |
| 1 | 5 | 4 | 6 | 2 | 4 | 5 | 1 | 1 | 3 | 2 | 1 | 1 | 5 | 40 |
| 1+ | 2 | | | | 2 | 2 | | 2 | | 2 | 1 | 2 | 5 | 18 |
| 2– | 6 | 1 | 6 | 1 | 1 | 2 | | 1 | 1 | | 6 | 7 | 1 | 33 |
| 2 | 10 | 1 | 10 | | 3 | 4 | 3 | 3 | 1 | 1 | 1 | 1 | 2 | 40 |
| 2+ | 10 | 3 | 4 | 1 | | 4 | 2 | 1 | 1 | 2 | | | 6 | 34 |
| 3– | 9 | 4 | 3 | 1 | 2 | 4 | 2 | | 1 | | | 2 | 3 | 31 |
| 3 | 10 | 1 | 7 | 2 | 1 | 7 | 3 | 3 | 12 | 2 | 1 | 5 | 3 | 57 |
| 3+ | 3 | 3 | 2 | | | 1 | 2 | 1 | | 2 | 1 | | | 15 |
| 4– | 1 | 1 | | | | 1 | 1 | | | | | | 2 | 6 |
| 4 | 13 | 3 | 9 | | 1 | 1 | 1 | 1 | 2 | 1 | | 3 | 2 | 37 |
| 4+ | 9 | 3 | 4 | | 1 | 1 | 1 | 1 | 5 | 1 | 1 | 4 | 6 | 37 |
| Total | 99 | 29 | 54 | 9 | 15 | 35 | 16 | 15 | 28 | 15 | 12 | 26 | 35 | 388 |

* Reading from left to right types of infection induced by Form 1 are given. Reading from top to bottom, types for Form 19 are given.

† The small rectangle includes those individuals which were as resistant as the parents to Form 1 and Form 19.

§ All flecked but no uredinia produced.

## DISCUSSION OF RESULTS

The study of the $F_2$ generation from a cross between Marquis (*Triticum vulgare*) and Mindum (*Triticum durum*) has shown clearly that all gradations of susceptibility and resistance to two biologic forms of *Puccinia graminis* may appear. Some evidence was obtained that susceptibility and resistance to Form 19 are governed by a single pair of genetic factors. No such evidence was obtained with Form 1. The appearance of transgressive types and of intermediates varying beyond the limits of the parents suggests the existence of several genetic factors.

Since the action of each biologic form was quite uniform on individual plants, it is safe to conclude that the urediniospores of each form probably were of the same genetic composition. Consequently the factors concerned in susceptibility and resistance must be located in the plants themselves. Further, since biologic forms differ in their parasitic capabilities on the same varieties of wheat, their genetic composition

cannot be the same. In all probability, then, factors governing susceptibility and resistance to different biologic forms are different in nature.

Stakman Parker and Piemeisel (10) have shown that the behavior of any given form is quite constant on pure line varieties of wheat. Given variety A, which is resistant to one biologic form but susceptible to a second, and variety B, which acts reciprocally to these two forms, these results show that a variety of wheat, which is resistant to both biologic forms, can be produced by crossing. This furnishes some hope that a variety which is resistant to all biologic forms eventually can be produced.

Although it has been demonstrated that plants resistant to more than one biologic form can be obtained by hybridization, we still are faced with the problem of breeding plants combining resistance and suitable agronomic characters, Hayes, Parker and Kurtzweil (3), by studying a cross between a resistant variety of *Triticum durum* and a susceptible variety of *Triticum vulgare*, have shown that when resistance occurs in the $F_2$ and $F_3$ generations it often is closely linked with durum characters. They demonstrated, however, that resistant, common-wheat types can be obtained. The problem is further complicated by the fact that crosses between durum or emmer (*T. dicoccum*) and common wheats often produce many sterile flowers. $F_1$ crosses made by the writer between Khapli (*Triticum dicoccum*), which is resistant to all known biologic forms, and Marquis (*T. vulgare*), have set no seed.

## SUMMARY

1. A study was made of the parasitic capabilities of two biologic forms of *Puccinia graminis* on the $F_2$ generation from a cross between *Triticum vulgare* and *T. durum*.

2. The parental plants, Mindum (*T. durum*) and Marquis (*T. vulgare*) react reciprocally to the biologic forms used.

3. The parasitic capabilities of the biologic forms previously had been studied carefully. Their action was known to be constant. Form 1 attacked Marquis normally but developed only very weakly on Mindum. Form 19, on the other hand, attacked Mindum heavily but developed only very weakly on Marquis.

4. Each $F_2$ plant was inoculated with both biologic forms of rust. The plants were inoculated with Form 19 when they were about 8 days old. After notes were taken the infected leaves were removed, and the plants were inoculated with Form 1.

5. Seedling plants were infected very easily. It was more difficult to infect older plants. This could not be attributed to increased resistance but only to the fact that older plants retained a film of moisture less easily than seedlings.

6. All gradations between complete susceptibility and immunity to both forms of rust appeared in the $F_2$ plants.

7. There was some evidence that a single main pair of genetic factors explained the manner of reaction to Form 19. Accepting this hypothesis, modifying factors must be present. These account for the various grades of infection obtained.

8. Infection results with Form 1 were not as satisfactory as those obtained with Form 19. There is no evidence that a single main pair of genetic factors explains the manner of reaction to Form 1.

9. All combinations of susceptibility and resistance of individual $F_2$ plants to the two biologic forms appeared. Out of a total of 388 plants, 35 were highly resistant to both forms of rust. This makes it reasonable to assume that varieties resistant to more than two biologic forms may be produced by hybridization. This fact, together with the demonstrated relative constancy of biologic forms, gives additional assurance that varieties of wheat, which will be commercially resistant over fairly extensive crop areas, eventually can be produced.

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# NOTES ON SAP STAIN FUNGI

ERNEST E. HUBERT

WITH PLATE VII AND FOUR FIGURES IN THE TEXT

Several samples of stained wood were collected by C. J. Humphrey at Metropolis, Ill., in connection with a study of decay in railroad ties (8). These along with other material were given to the writer for examination to determine the action of the stain fungi on the tissues.* The fungi studied included *Lasiosphaeria pezizula* (B. & C.) Sacc. and *Ceratostomella spp.*, the former on beech (*Fagus atropunicea* (Marsh.) Sudw.), red gum (*Liquidambar styraciflua* Linn.), and persimmon (*Diospyros virginiana* Linn.), the latter on white pine (*Pinus strobus* Linn.), scrub pine (*P. virginiana* Mill.), western white pine (*P. monticola* Dougl.), western yellow pine (*P. ponderosa* Laws.), shortleaf pine (*P. echinata* Mill.), loblolly pine (*P. taeda* Linn.), red gum, persimmon, aspen (*Populus tremuloides* Michx.) and northern white cedar (*Thuja occidentalis* Linn.). Railroad tie material is represented by red gum, persimmon, beech, and loblolly pine.

The safranin and lichtgrün staining process as described by Colley (3) was used in preparing the slides. This staining method aided in the study of the young hyaline hyphae in the wood tissues.

## LASIOSPHAERIA PEZIZULA

As a sap-staining organism *Lasiosphaeria pezizula* has been previously reported by Humphrey (8). Ellis and Everhart (4) give a brief description of the fungus under the name *Herpotrichia pezizula* B. & C., and state that it occurs on decaying wood and bark. No other references have been found regarding the wood-inhabiting nature of this Pyrenomycete.

The stain produced in wood by the hyphae of this fungus resembles somewhat the blue stain in sapwood caused by the species of Ceratostomella, but differs in that it is of a grayish olive instead of a grayish blue (Payne's gray) color (11). The stain, which penetrates more readily the cut ends, extends unevenly into the wood for some distance, usually one to four inches, and is found principally in the sapwood, although it is not uncommon to find the heartwood stained. The stain is undoubtedly produced by the massing of the hyphae in the tissues and the more intensely stained sections are found to contain the greater concentration of hyphae. No evidence was found that the color was due to a staining of the cell walls or to the presence in the cells of colored infiltrations or by-products.

* Thanks are due P. V. Siggers, for aid in the examination of tie material.

Under the microscope the older mature hyphae in wood (Fig. 1A) are olivaceous in color and can be distinguished from the dark brownish hyphae of the blue-stain fungi here studied. The hyphae are usually much twisted and the individual cells appear to be somewhat shorter than those in the *Ceratostomella spp.* studied. An interesting spore-like formation (Fig. 1E and plate VII, fig. 3) is found within the tissues near the surface of the infected wood. These formations are produced from the older, large, olive-colored hyphae and are found in the cells near the surface or are present upon the surface of the wood along

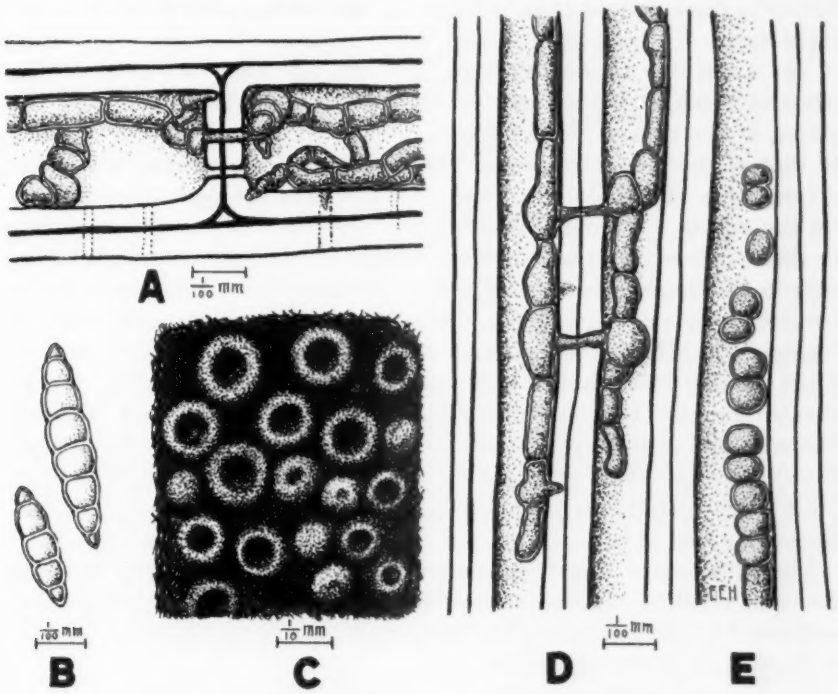


FIG. 1. LASIOSPHAERIA PEZIZULA, A WOOD-STAINING FUNGUS.

*A*. Mature hyphae of *Lasiosphaeria pezizula* Sacc. in ray cells of persimmon wood. Penetration of cell walls by way of the simple pits. Radial section; *B*. Ascospores of *Lasiosphaeria pezizula; C*. Perithecia of *Lasiosphaeria pezizula* on persimmon wood. Those showing no sunken areas are in the early state of development. The older ones collapse and the sunken areas resemble minute craters; *D*. Mature hyphae of *Lasiosphaeria pezizula* in the wood fiber cells of persimmon showing penetration of the tangential wall. The tips of two hyphal threads are seen approaching the middle lamella. Radial section; *E*. An apparent secondary spore formation found a short distance in from the surface of the wood. *Lasiosphaeria pezizula* in persimmon wood. Radial section.

with the small, black perithecia (Fig. 1C).

The young hyphae are much smaller when young than when mature, and are hyaline. Differential staining is often needed to bring the

young hyphae into clear vision. The surface of the wood infected with this organism usually develops a layer of olivaceous-black mycelium with occasional clusters of small closely grouped perithecia, which when old collapse and resemble minute craters (Fig. 1C). The color and shape of the perithecia and the one-to-seven-celled ascospores they contain (Fig. 1B) readily distinguish this fungus from the blue-stain fungi. The latter have small black flask-shaped perithecia, with long or short beaks, containing minute, unicellular hyaline spores in asci which disappear soon after maturity. The superficial growth of the former is more matted than is usual for the surface growth of the blue-stain fungi.

The stain due to *Lasiosphaeria pezizula* has been observed in the wood of beech, red gum and persimmon cut for railroad ties, and the action of the hyphae within the tissues of these hosts is very similar to that of the blue-stain fungi. The majority of the hyphae are found in and near the medullary rays, and the rays are somewhat altered by the attack. Decomposition of the ray cells comparable in extent to that produced by the blue-stain fungi has not been observed. New cells in the neighborhood of the rays are usually invaded through the bordered pits and the simple pits. Penetration of the cell walls of tracheids is often observed and the hyphae are always constricted when passing through the bore holes (Fig. 1D). This indicates that penetration was effected when the hyphae were young and of small diameter and also suggests that the young growing tips of the hyphae possess the necessary enzymes for the dissolution of the lignified cell walls. The demonstration of young hyphae in the process of penetration is proof of this.

The presence of this fungus in wood does not necessarily indicate that the wood is undergoing serious decay, but wood-destroying fungi may also be expected to develop since conditions favorable to the growth of the one also favor the other.

### CERATOSTOMELLA SPP.

The common defect produced in "sap" lumber by the species of Ceratostomella is called "blue-stain." Boards so stained are graded lower than unstained boards, and since much of this staining develops in otherwise high grade material, considerable attention has been directed to the study of the blue-stain fungi. Very little work has been done to determine the effect of the blue-stain fungi on the walls of the invaded cells. The blue stain of pine wood caused by a species of Ceratostomella was first noted in Europe by Hartig (5, 6) in 1878.

In this country von Schrenk (13) in 1903 studied in considerable detail the effect produced on the wood tissues by *C. pilifera* (Fr.) Wint. His drawings indicate that the fungus can readily attack and dissolve the walls between the cells of the medullary rays of pine wood. The

inner walls of the rays are also shown to be affected, being thin and evidently weakened. He states: "The hyphae are apparently able to puncture the unlignified walls here and there, but they stop at that point."

Münch (9) has brought out some interesting points concerning the blue-stain of coniferous woods. He states that "the blueing fungi do not attack the wood substance (cell walls) to any great extent but live upon the contents of the parenchyma cells." He finds that these fungi can also become parasitic in standing living trees under the conditions

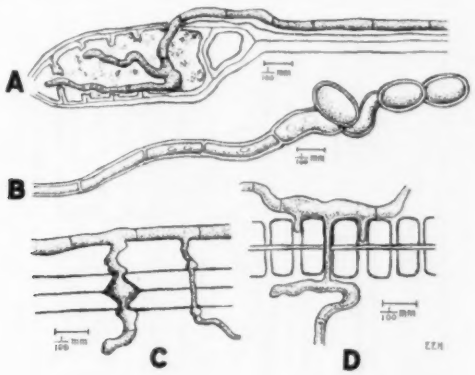


FIG. 2. BLUE STAIN OF WOOD

*A*. Decomposition of medullary ray cells in the wood of shortleaf pine by hyphae of the blue-stain fungus. Note the effect on the cell wall adjacent to the older portion of the hyphal thread; *B*. Terminal hypha of *Ceratostomella sp.* with spore-like cells in the wood of loblolly pine. This formation occurs but rarely; *C*. Hyphae of the blue-stain fungus in pine wood. Two penetrations through tracheid walls, one by way of the bordered pit the other by boring through the cell wall; *D*. Hyphae of the blue-stain fungus in persimmon wood penetrating the wall of a ray cell through the natural openings. Tangential section.

of his experiment provided the wood contains sufficient air and little water. In the above trees he had chopped off part of the roots and had sawed through cortex and sapwood, thus partly girdling the tree. The stain appeared only between the two saw cuts. The mycelia of two of these fungi, *C. pini* Münch and *C. coerulea* Münch, he found were capable of killing the living cells of coniferous wood and in part that of hardwoods. His figure (9, fig. 2, p. 542) shows the penetration of tracheid cell walls of pine by *C. pini*. He finds these penetrations rarer than those through the bordered pits and disagrees with von Schrenk's statement (13) that "unlignified membranes, that is, membranes consisting of cellulose" are decomposed.

Hedgcock (7) studied the fungi accompanying various stains in wood and found several species of *Ceratostomella* causing blue-stain in both coniferous and broadleaf hosts.

In the blued wood of the hosts examined for this study it was found that quite frequently the hyphae penetrated the tangential cell walls of tracheids and fibers which were not in contact or adjacent to the medullary rays (Figs. 2C, 3 and 4). In one case where the sap wood of scrub pine was found heavily blued, the examination of radial sections showed a single young hypha penetrating three tracheid cell walls

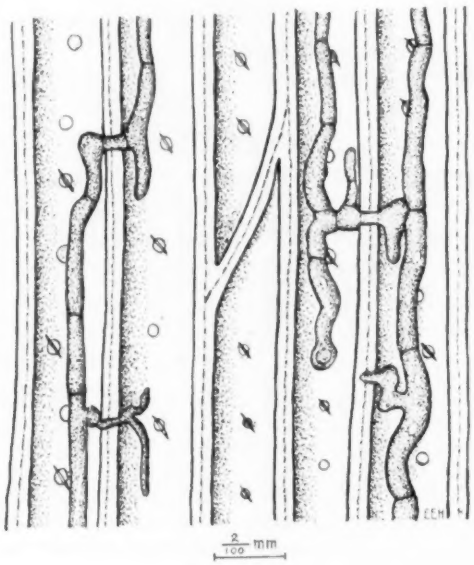


FIG. 3. CERATOSTOMELLA SP.

Hyphae of the blue-stain fungus in persimmon wood showing penetration through fiber cell walls. Radial section drawn with camera lucids.

(Fig. 4). In radial sections of blued shingles of northern white cedar as high as 64 tracheid cell walls were observed penetrated by a single hypha (Plate I, fig. 1). The hyphae are considerably constricted where they pass through bore holes in the tracheid walls and swellings occur in the hyphae at the points of entrance and emergence (Plate I, figs. 2 & 4). The young growing tip of the hypha apparently is the source of the enzymes which dissolve the cell walls, since the bore holes are nearly always of diameter corresponding to the size of the young hyphae. An exception to this is found in persimmon wood where the fungus apparently is able to enlarge slightly the bore holes first formed

by the young hyphae (Fig. 3), since a variation in the diameter of the bore holes and amount of constriction is noticeable. It seems reasonable to suppose that any growing tips of young hyphae which are developing in contact with the walls and parallel to the long axes of the tracheids or fibers would be capable of secreting enzymes and thus affect the tertiary walls (Fig. 2A).

In the pines and persimmon it was frequently observed that the tracheid or fiber cell walls in close contact with certain hyphae were much thinner than the adjacent cell walls which had no hyphae in contact (Fig. 2A).

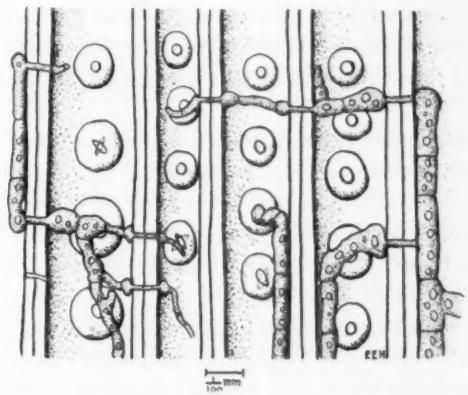

FIG. 4. CERATOSTOMELLA PILIFERA.

Hyphae of the blue-stain fungus *C. pilifera* in the wood of *Pinus virginiana* showing penetration of young hyphae through the walls of tracheid cells. One hypha has penetrated three cell walls. Radial section. Drawn from fresh material with camera lucida.

More often the hyphae search out the simple and bordered pits and pass through these, the shape of the fungous thread conforming to the shape of the opening (Fig. 2C, plate I, fig. 5). In the case of simple pits, the hyphae must dissolve the middle membrane in order to pass through. In the bordered pits of coniferous wood the perforated torus membrane (1, 2)[1] forms but a weak barrier, yet a certain amount of tissue decomposition is necessary to enlarge sufficiently such minute perforations as already exist.

In the less lignified medullary ray cells the effect of the fungus attack is very pronounced. The cell walls between the ray cells are completey decomposed, the radial walls of the ray becoming very thin and broken, so that there is less than a shell or framework of the ray left (Fig. 2A).

The medullary rays, particularly, present a seriously decomposed and evidently weakened aspect, which, when multiplied for the entire piece of infected wood, point to the possibility of a loss in strength to a certain extent. For ordinary uses blued wood is apparently as strong as unstained wood (14). Few data are at hand to indicate that the stained wood is below normal in strength.

In the study of blue-stain in aspen, red gum, white pine, scrub pine, and northern white cedar, fragments of the blued wood were successfully cultured on malt agar media. In some of these woods the fungus had previously been dormant for a considerable length of time. Successful cultures were also secured from fragments of blue-stained wood taken from the sap zone of a piece of structural timber which had been in use in a factory building for seven years. The timbers in this building were shipped "green" from the mill and put in place during a rainy period.

The above observation led to a search for the origin of the new hyphae produced in such cultures. Radial sections were cut from a heavily blued white pine block which had been kept in the dry air of the laboratory for more than a year. These were washed twice in distilled water and placed on a thin layer of malt agar which had previously been spread on a sterile glass slide. A sterile cover glass was pressed firmly over the sections. Examination under the microscope at regular intervals showed hyaline hyphae radiating from the old brown masses of anastomosed blue-stain hyphae in the medullary rays. Closer examination showed these new hyphae issuing directly from portions of the old hyphae. The exact process by which the new hyphae are developed from the old is as yet undetermined but observations so far indicate that growth from the broken end of the old hypha often takes place. Many of the new hyphae were observed extending outward from the side walls of individual cells of the old hyphae. The new hyphae developing on malt agar are much larger, more guttulate and develop more rapidly than those grown by the above method in distilled water. In many of the cover-glass cultures the new hyphae produce abundant conidia which resemble closely in size, form and manner of development those described and illustrated by Münch (9) as belonging to *C. pini*. In the agar cultures the hyphal mats were densest in the media immediately surrounding the air spaces inclosed in the agar between the slide and cover glass. This gives an indication of the air requirement of the fungus in the presence of abundant moisture (9).

As a preliminary step to determine the effect the Ceratostomella hyphae have upon the cell structure, microscopic sections were treated with the following reagents and the color reactions noted. The phloroglucin, aniline sulphate, and para-nitroaniline reactions indicated no

apparent change in the lignin complex of the cell wall. The para-nitroaniline produces a bright orange yellow color in lignified tissues and serves to accentuate the bore holes. It furnishes an excellent, clear-cut contrast between lignified wood cells and wood cells with the lignin complex removed. A few of the standard reagents for cellulose were tried on the blued sections but furnished no information of value.

In blued pine wood peculiar spore-like cells were observed in two cases (Fig. 2B). These cells appeared at the tips of old, mature hyphae, growing in the tracheids. Nothing definite can be stated regarding these formations and further observations are necessary to determine their origin and their function.

Considerable work has been done to determine the effect of the blue-stain fungi upon the mechanical properties of the wood they discolor. A variety of results were obtained by the various workers. Rudeloff (12) found that the compressive strength of wood infected with a blue-stain organism did not differ from unstained wood, but it appears he took no account of the variation in moisture content of the test pieces (9), and the results are therefore not reliable. Von Schrenk (13) gives a series of tests made with stained and unstained pieces stating that for all practical purposes the stained wood is as strong as the unstained. He further notes that the higher results obtained for the stained wood in tests for compression parallel and perpendicular to the grain is probably due to the difference in moisture content between the stained and unstained pieces. He observed a greater resistance to splitting along a tangential plane in the blued wood than in the unaffected wood. Münch (9) tested pine wood infected for six months with the blue-stain fungus, *C. pini*, and found that the wood became "slightly lighter and weaker with regard to compression." These effects he attributed to the presence of other fungi in the test pieces. His general conclusion (9, p. 319) is that "neither the specific gravity nor resistance to compression are altered through blue-stain." Weiss and Barnum (14) tested stained and unstained pieces of longleaf pine (*P. palustris*) and short-leaf pine (*P. echinata*) and concluded that for all practical purposes the blued wood was as strong as the green (unstained) wood. But the heavily stained short-leaf pine having the same moisture content was found to be "slightly weaker, less tough, and showed less surface hardness than the unstained." The St. Louis tests (13) on stained and unstained yellow pine (*P. ponderosa*) gave higher results for the stained wood in compression parallel and perpendicular to grain than the unstained wood. Weiss and Barnum (14) stated that this result was probably due to the fact that the stained wood was drier than the normal wood with which it was compared.

Such reliable strength tests as have been made indicate a slight weak-

ening effect due to the blue-stain. Since decay-producing fungi may develop simultaneously with blue-stain in the same timber, this furnishes a basis for discriminating against the use of blued wood for structural purposes where the failure of the piece is likely to cause loss of life and property. For this reason blued wood and wood stained by *L. pezizula* should be rejected in airplane construction unless careful tests indicate otherwise.

A few cases have been noted where blued wood steamed and bent for use in the manufacture of furniture has failed under the bending process. It would be of value to determine whether the blue-stain fungus in the broken bent material is the cause of the failure or whether it is due to wood-destroying fungi accompanying the blue-stain. These and related problems need solution.

It is apparent from the foregoing citations that the moisture content of the test piece appreciably affects the results. That the variation in moisture content of wood greatly affects the test data is well known to engineers in timber mechanics, as the following quotation attests, "a one per cent change of moisture causes a four per cent change of modulus of rupture" (10, p. 18) when the wood has a moisture content of twelve per cent. The degree of comparative equality of the matched sound and infected pieces in itself involves a large number of variable factors and the task of detecting the slight weakening effects produced in wood by fungi becomes more complex and difficult when one considers that, in testing, a variation of from seven to sixteen per cent from the average for normal sound wood of any random tree of the same species may occur (10).

The fungus producing blue stain in the pieces of *Pinus strobus* and *Pinus virginiana* examined in this study resembles closely, with exceptions as noted, *Ceratostomella pilifera* (Fr.) Wint. (7) and no doubt belongs in the *C. pilifera* group as defined by Münch (9).

## SUMMARY

Information regarding two stains found in sap-wood of commercial woods is presented.

The stain produced by *Lasiosphaeria pezizula* is usually grayish-olive in color and is found both in sap-wood and in heartwood. The stain produced by species of Ceratostomella is grayish-blue in color and is confined almost entirely to the sapwood.

The perithecia of *L. pezizula* are minute, black bodies, spherical when young and collapsed when old, resembling minute craters. These are gregarious upon a mat of olivaceous-black hyphae resting upon the surface of the wood. The blue stain fungi produce minute flask-shaped perithecia with beaks of various lengths.

The hyphae of *L. pezizula* and *Ceratostomella spp.* penetrate the walls of the wood fibers and tracheids. It is apparent that these fungi affect the cell walls of their hosts to a certain extent.

The blue-stain hyphae, dormant in the wood for a considerable period of time, are capable of reviving and sending forth new hyphae on the return of favorable growth conditions. The new hyphae usually originate from the old, anastomosed hyphae found in the medullary rays, and are influenced in their development by the media in which they are growing and the amount of available air within the media.

Such reliable strength tests as have been made indicate a slight weakening effect due to blue-stain caused by *Ceratostomella spp.*

The moisture content of test pieces plays an important part in the testing of matched sticks. Some of the test work on stained and unstained wood in which this factor was not controlled must for this reason be disregarded.

The slight weakening due to blue-stain, is in itself of no special importance in larger material, but it serves to hide the effect produced by wood-destroying fungi which often accompany it. In this way it may also be found to affect wood of smaller dimensions to an appreciable extent. The failure of blued wood while undergoing steam bending in the process of the manufacture of furniture and other products suggests a new field of investigation which has a very practical aspect.

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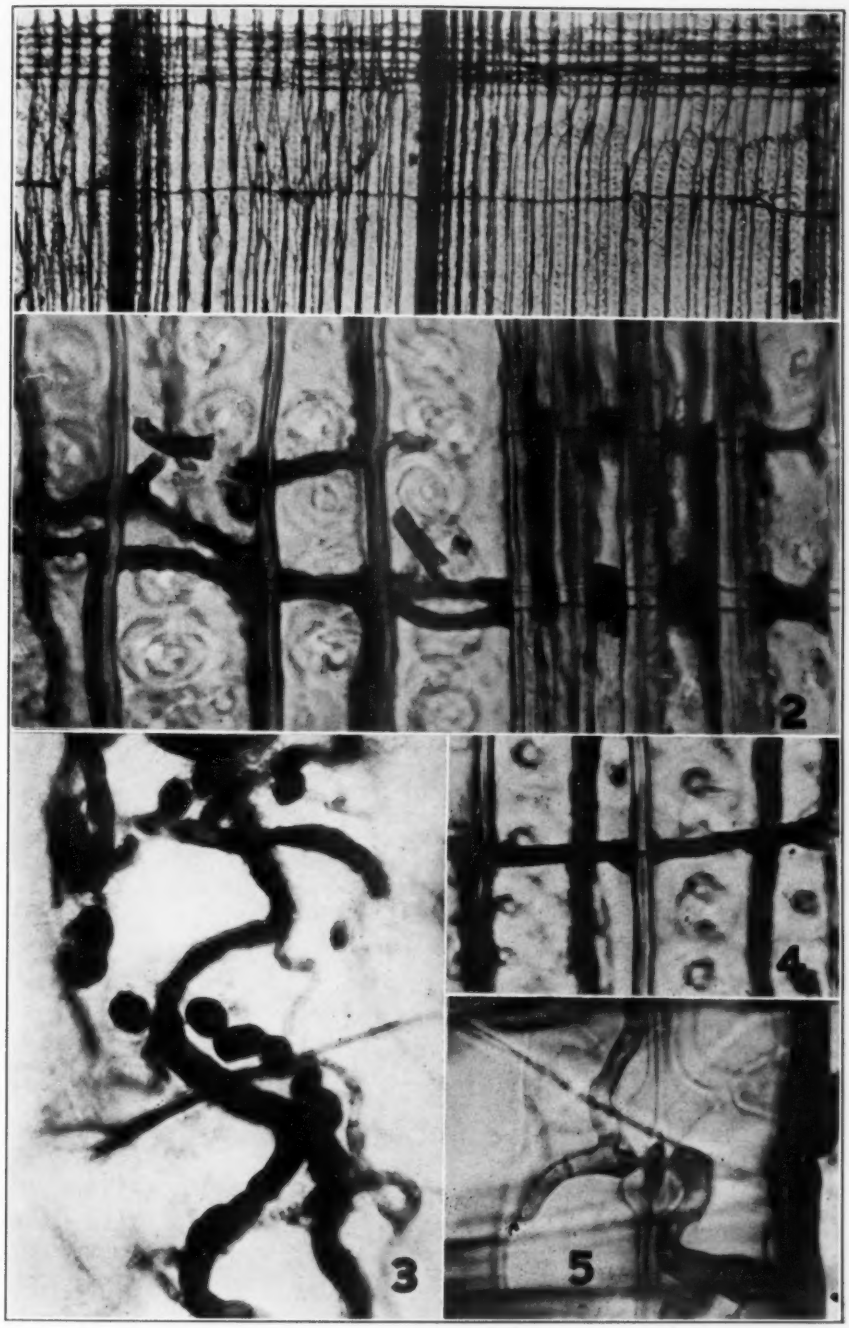
PLATE VII. WOOD STAINING BY FUNGI

Fig. 1. Photomicrograph showing a hypha of the blue-stain fungus penetrating several tracheid walls in northern white cedar. Radial section stained with safranin; Fig. 2. The extreme right-hand portion of Fig. 1 enlarged showing the constricted hyphae in the bore holes made in the tracheid walls; Fig. 3. Photomicrograph showing spore-like development of hyphae of *Lasiosphaeria pezizula* in persimmon wood. Radial section stained with safranin and lichtgrün; Fig. 4. Enlarged portion of the hypha shown in Fig. 1. Note the swelling of the hypha at entrance and emergence and the constriction where passage is secured through the cell wall; Fig. 5. Photomicrograph showing hypha of the blue-stain fungus passing through a bordered pit in the wood of loblolly pine. Tangential section stained with safranin and lichtgrün. Photograph by P. V. Siggers.




FUNGI STAINING SAP WOOD

# TWO NEW SCLEROTINIA DISEASES

B. F. Dana

With Plate VIII

A Sclerotinia disease of *Amelanchier cusickii* has been observed and studied by the writer[1] as it occurs on the campus of the State College of Washington and in the vicinity of Pullman.

The first symptoms of the disease are manifested in a leaf blight which attacks only young and immature leaves. Only a small percentage of the leaves are involved, the affected portions turning black at first and later brown as the tissue dries out. The margins of affected leaves roll upward, sometimes making them more or less tubular. On the inner surface of such leaves, and especially along the midrib of all affected leaves, is produced a grey, powdery layer of spores of the Monilia stage of the fungus. Occasionally a leaf is found which is only partially affected due to late infections of the more mature leaves. The leaf blight, as it claims only a small portion of the leaves, is not especially detrimental to the host plant.

Following the leaf blight the disease appears on the half-grown fruits, and in most cases all the fruits of a cluster are involved. They dry and hang on the twig until the end of the growing season. The fungus does not pass into the twig either from the leaf or from the blighted fruit. In the fall, diseased fruits fall to the ground and persist throughout the winter as mummies.

Careful microscopic and cultural study of the fungus causing this disease has shown it to be a species of Sclerotinia. The stage on the leaves is a true Monilia and is produced in considerable abundance. The fungus has not been observed to produce spores on the fruits. The conidia from the leaves are lemon-shaped to globular in form. Repeated isolations from diseased leaves have been made and the fungus obtained in pure cultures which remained sterile with the exception of microconidia produced in old cultures. The microconidia are globular, continuous, homogenous, and are produced singly on non-septate sporophores. The sporophores, one to several to each cell, are borne radially on prostrate hyphae, their position showing no constant relation to the septa.

[1]Studies here reported are an abridgement of work submitted to the faculty of State College of Washington as a thesis for the Master's degree in 1917. Acknowledgment is due to Dr. F. D. Heald, under whose direction the work was carried on, for helpful suggestions, criticisms and assistance in providing illustrations.

The ascigerous stage of the fungus is produced in great abundance on the fallen over-wintered fruits. Because of failure to obtain germination of ascospores no cultural proof can be offered that this is the ascigerous stage of the fungus on leaves and fruits. The abundance of this stage on the over-wintered fruits under bushes having much of the fruit blight the previous season is considered sufficient proof that the two stages are connected. During the seasons when observations were made, the ascigerous stage was first found after the disease had appeared on the leaves. This would raise the question as to what the real role of the ascigerous stage is in the life history of the parasite, a question the available data do not answer. The apothecia are light grey and are fairly uniform in size, the majority producing disks 1–3 millimeters in diameter. The average diameter of those apothecia observed to be discharging spores was 2–3 millimeters. From one to many apothecia with asci of the typical Sclerotinia type are formed on each mummy, the largest number observed being forty-seven. The majority of the ascospores measure 2–3 × 8–10 microns.

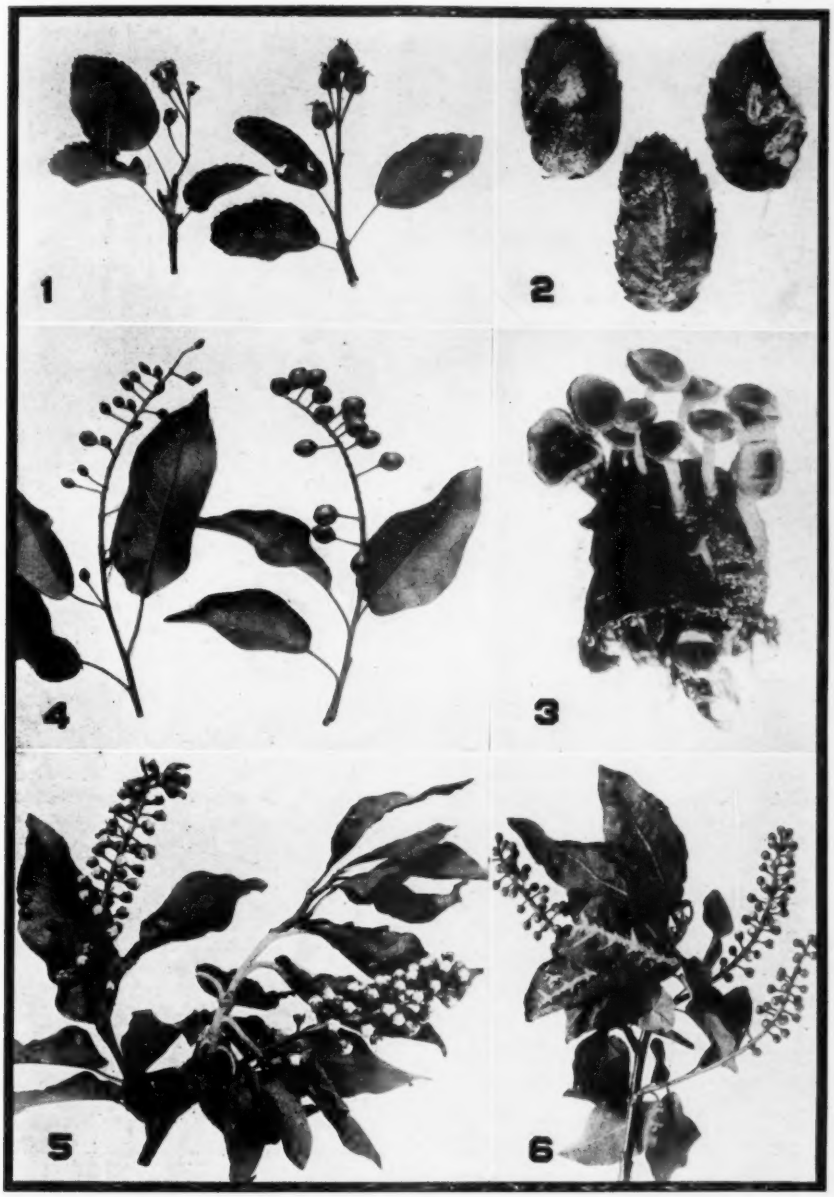
A study was made of the descriptions of the species of Sclerotinia reported on the wild and cultivated pome fruits. But one species was listed on the Amelanchier and no description was given of an ascigerous stage. Measurements of conidia, asci, and ascospores of the various species do not agree with those of the Sclerotinia on the Amelanchier in the Palouse Country, the asci and ascospores particularly of this species being very much smaller than for other species described. The name *Sclerotinia gregaria* is proposed and the following description is given:

*Sclerotinia gregaria* nov. sp.

Apothecia gregarious, pale gray in color at first, later becoming darker. Disk, at first cup-shaped, later flatter, circular, 1–5 mm. diameter. Stipe 1–3 mm. in length. Asci club-shaped, 5–6 × 48–57 microns, eight-spored, spores in upper half. Ascospores uni-seriate, hyaline, biguttulate, pointed at ends, 1.8–3.5 × 8–12 microns. Paraphyses simple or branched, 2 × 32 microns. Conidia light gray in mass, lemon-shaped to globular, 4–13 × 5–13 microns, not produced in culture. Microconidia from cultures are globular, homogenous, produced singly on short sporophores, 2–3.5 microns, the majority 2.5–3.0 microns.

Habitat: Conidial stage, on living leaves and fruit of *Amelanchier cusickii;* ascigerous stage, on over-wintering mummies of *A. cusickii.*

Type specimens collected at Pullman, Washington, May 1, 1916 (No. 250); June 23, 1916 (No. 294); and May 19, 1916 (No. 295). Numbers refer to Plant Pathology Collections, Washington Agricultural Experiment Station.




SCLEROTINIA GREGARIA AND S. DEMISSA

*Sclerotinia gregaria* on *Amelanchier cusickii*, Fig. 1. Diseased fruits (left, normal fruits (right). Fig. 2. Spore production increased by incubation in damp chamber. Fig. 3. Overwintered fruit showing production of apothecia.

*Sclerotinia demissa* on *Prunus demissa*, Fig. 4. Diseased fruits (left), normal fruits (right). Fig. 5. Blighting of shoot. White color due to spore production. Fig. 6. Leaf blight showing natural production of spores along midrib.

A disease of *Prunus demissa* also due to a species of Sclerotinia was studied along with the similar disease on the *Amelanchier* just described. This disease is very prevalent each year in the vicinity of Pullman, doing considerable injury to the host.

Soon after the shrub leaves out in the spring and while the foliage is yet tender, the first stage of the disease appears in the form of a leaf and twig blight. The young leaves are invaded and turn brown, roll slightly and hang to the plant until late in the summer. From the leaf the disease often passes to the twig and causes extensive blighting of the shoot. Gumming often takes place in the twig lesions. Girdling of the twig and the death of the outward portion is of frequent occurrence. Fruits are attacked when about half grown. They cease growing, turn brown and soon fall. Only a part of the fruits in a cluster are generally attacked.

The fungus causing this disease is also a typical Sclerotinia. The Monilia stage is produced in profusion on diseased leaves and twigs and on the leaves persists as a gray powdery layer. No spore production has been observed on the fruits. Conidia direct from the host are usually lemon-shaped, but also globular, the majority being 6 × 8 microns, but those from cultures are smaller. From a cultural study it was learned that the conidia are produced in unbranched chains from short conidiophores distributed laterally on the vegetative hyphae. The sporophores are at first simple but later may become septate and branched, each branch bearing a chain of spores.

The ascigerous stage arises from fallen over-wintered fruits and is mature some time before the leaves reach the susceptible stage. The brown, glabrous apothecia are produced singly or in pairs. The disk measures 2–10 mm., depending on maturity largely. The asci are hyaline, eight-spored, blunt at the tip and are interspersed by paraphyses. Ascospores show one end more pointed than the other with the majority 5 × 12 microns.

Cultures were readily obtained from leaves and fruits but no isolations were successful from the ascospores. Mature apothecia were allowed to discharge spores on agar plates but no germination of ascospores was obtained on three per cent dextrose agar. The season of the ascigerous stage was over before other media could be tried.

Nine species of Sclerotinia have been described on wild and cultivated species of Prunus, some of which are very closely related to *Prunus demissa*. Descriptions in many cases were unsatisfactory but such descriptions as were given did not agree with the *Sclerotinia* found on *P. demissa*, consequently the name *Sclerotinia demissa* is proposed and the following technical description given:

*Sclerotinia demissa* nov. sp.

Apothecia mostly solitary, sometimes two from a mummy, long stipitate, brown, glabrous, at first cup-shaped, later flatter. Disk circular, 2–10 mm. in diameter. Stipe 1–3.5 cm. in length. Asci hyaline, eight-spored, with blunt ends, 7–4 × 150–160 microns. Ascospores elliptical, uni-seriate, hyaline, one end commonly more pointed than the other, 5–6 × 9–15 microns. Conidia, oval to globose, hyaline, cream-colored in mass, continuous, 3–9 × 7–14 microns, produced in unbranched chains. Habitat: *Conidial stage*, on living leaves, twigs, and fruits of *Prunus demissa; Ascigerous stage*, on over-wintering mummies of *P. demissa*.

Type specimens collected at Pullman, Washington, May 5, 1916 (No. 255); May 17, 1916 (No. 267); June 23, 1916 (No. 290); and June 28, 1916 (No. 313). Numbers refer to Plant Pathology Collections, Washington Agricultural Experiment Station.

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*Printers strike.* The members of the American Phytopathological Society doubtless know that a national strike of printers has been in progress since early May. This is the cause of the great delay in publication of Phytopathology for April and the succeeding numbers. It is hoped to make up the lost time very shortly as operations are now being resumed.